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Search History

DATE: Wednesday, September 04, 2002 Printable Copy Create Case

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	DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ		
L10	L6 and l8	20	L10
L9	l3 and L8	2	L9
L8	herbicid$6	64149	L8
L7	l3 and L6	0	L7
L6	l4 or L5	54	L6
L5	formyl adj2 ((hydroxyamino adj propylphosphon$3) or hydroxyaminopropylphosphon$3)	17	L5
L4	fosmidomycin	43	L4
L3	l1 or L2	492	L3
L2	amino adj2 hydroxybutylidene adj3 bisphosphon$3	114	L2
L1	alendron$3	456	L1

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L9	l6 and L8	0	L9
L8	squalene	3907	L8
L7	l1 and L6	0	L7
L6	Clomazone or ((chlorobenzyl or chlorophenylmethyl or (chlorophenyl methyl)) adj3 ((dimethyl adj3 oxazolidin$3) or dimethylisoxazolidin$3)) or fenoxan or dimehazone or gamit or magister or fmc57020 or (fmc 57020)	660	L6
L5	l1 and L2	6	L5
L4	l1 same L2	0	L4
L3	l1 with L2	0	L3
L2	herbicid$5	68230	L2
L1	squalene synthase	320	L1

END OF SEARCH HISTORY

L5: Entry 1 of 6 File: PGPB Mar 21, 2002

DOCUMENT-IDENTIFIER: US 20020035058 A1
TITLE: Isopentenyl pyrophosphate isomerase (IPI) and/or prenyl transferase inhibitors

Summary of Invention Paragraph (26):
[0026] Attempts have therefore been made to block cholesterol synthesis more specifically by using inhibitors of squalene synthase. This enzyme catalyzes the reductive dimerization of farnesyl pyrophosphate to form squalene at the final branch point of the cholesterol biosynthetic pathway (see FIG. 1). Since the enzyme is unique to this pathway, selective inhibitors of squalene synthase might be expected to reduce cholesterol levels without eliminating the pool of essential branch products of the isoprene pathway (such as dolichol, coenzyme-Q and prenylated proteins). Accordingly, it has been suggested that squalene synthase inhibitors could have advantages over the statins as hypolipidaemics. On the basis of this rationale, several different types of squalene synthase inhibitors have been isolated or synthesized (Ciosek et al. (1993) J. Biol. Chem. Vol. 268 (33), pages 24832-24837 and Amin et al. (1992) J. Lipid Res. Vol. 33, pages 1657-1663). Some of the known squalene synthase inhibitors are lipophilic bisphosphonates.

Summary of Invention Paragraph (28):
[0028] Thus, the squalene synthase inhibitors may not be as effective as the statins in alleviating cardiovascular disease attendant on hyperlipidaemia, and may be less effective in lowering cholesterol levels.

Summary of Invention Paragraph (55):
[0055] For example, the IPI and/or prenyl transferase inhibitors of the present invention find particular utility in the modulation of lipid metabolism, cell proliferation, isoprenoid-related cellular apoptosis and cellular signal transduction. The IPI and/or prenyl transferase inhibitors are also useful as fungicides or herbicides. The inhibitors may have dual inhibitory activity, and inhibit both IPI and prenyl transferase. They may also inhibit other isoprenoid synthetic enzymes.

Summary of Invention Paragraph (59):
[0059] The site of action of the IPI and prenyl transferase inhibitors of the present invention is shown in FIG. 1, along with those of the known HMGCoA reductase and squalene synthase inhibitors. It can be seen that the IPI and/or prenyl transferase inhibitors of the present invention act at a point in the cholesterol biosynthetic pathway before the synthesis of farnesyl pyrophosphate. In contrast, the known squalene synthase inhibitors act after the synthesis of farnesyl pyrophosphate. Thus, like the statins, the IPI and/or prenyl transferase inhibitors of the invention are upstream inhibitors, in that they inhibit both the cholesterol biosynthetic pathway and the various isoprenoid pathways.

Summary of Invention Paragraph (97):
[0097] When administered in suitable concentrations, the IPI and/or prenyl transferase inhibitors of the present invention can also be used to block sterol synthesis in plants and fungi. They therefore find utility as fungicides and/or herbicides.

Summary of Invention Paragraph (145):
[0145] The enzyme to be selectively inhibited is preferably selected from the

enzymes squalene synthase, protein prenyl transferase, cis-prenyl transferase and trans-prenyl transferase (geranylgeranylpyrophosphate synthase).

Detail Description Paragraph (24):
[0182] The exact enzymes of the mevalonate pathway that are inhibited by bisphosphonates remain to be identified. Mevastatin is an inhibitor of HMG-COA reductase and thus prevents synthesis of mevalonate. Hence, mevastatin-induced apoptosis could be prevented by addition of mevalonic acid lactone or the mevalonate-derived compounds FPP and GGPP. By contrast, addition of either FPP or GGPP, but not mevalonic acid lactone, partially prevented alendronate-induced apoptosis. Hence, alendronate appears to inhibit enzymes later in the mevalonate pathway than HMG-CoA reductase. It is possible that both FPP synthase and GGPP synthase are inhibited, since bisphosphonates appeared to prevent the incorporation of mevalonate into FPP and GGPP. However, since FPP is itself converted to GGPP by GGPP synthase, it is also possible that the inhibitory effects of bisphosphonates on both farnesylation and geranylgeranylation of proteins could be the result of inhibition of FPP synthase alone. Alternatively, BPs could prevent the transfer of prenyl groups to proteins by prenyl protein transferases. It is perhaps more likely that bisphosphonates (like .alpha.-hydroxyfarnesylphosphonate) can actually inhibit several enzymes of the mevalonate pathway that contain similar prenyl-pyrophosphate binding sites. This is supported by the findings of Amin et al, since YM175 and ibandronate (but not alendronate) were potent inhibitors of squalene synthase, whilst we have demonstrated that YM175 and ibandronate probably also inhibit enzymes involved in FPP and GGPP synthesis. The potency of bisphosphonates could therefore depend on the combination of enzymes that are inhibited.

CLAIMS:

13. A herbicide or fungicide comprising an IPI and/or prenyl transferase inhibitor.

14. Use of an inhibitor of IPI and/or prenyl transferase as a herbicide or fungicide.

L4: Entry 3 of 43 File: PGPB May 16, 2002

DOCUMENT-IDENTIFIER: US 20020058321 A1
TITLE: Method of determining the activity of 1-deoxy-D-xylulose-5-phosphate reductoisomerase and 1-deoxy-D-xylulose-5-phosphate synthase

Summary of Invention Paragraph (6):
[0006] This is why the 1-deoxy-xylulose-5-phosphate biosynthetic pathway is of particular interest in the search for new herbicidally active compounds. In particular the two enzymes 1-deoxy-D-xylulose-5-phosphate synthase (DXPS) and 1-deoxy-D xylulose-5-phosphate reductoisomerase (DXPR) are of central importance. It has already been demonstrated that DXPS (CLA I) is essential for the development of a normal plant (Mandel et al (1996), Plant J. 9, 649-658). This discovery supports the expectation that a herbicidal compound which affects DXPS activity has a herbicidal action. Also, it has been demonstrated that bacterial DXFR is inhibited by the herbicidal compound Fosmidomycin, which is already known (Zeidler et al. (1998), Z. Naturforsch. 53, 980-986; Kuzuyama et al. (1998), Tetrahedron Lett. 39, 7913-7916). However, there are no commercially useable herbicides, which affect DXPS or DXPR activity. In the search for new, improved herbicides, both enzymes are therefore of high importance as sites of action. The 1-deoxy-xylulose-5-phosphate biosynthetic pathway also has importance in microorganisms, especially in parasitic microorganisms as, for example, bacteria or plasmodia. The treatment of infectious diseases, in particular the treatment of malaria, may be based on the inhibition of this metabolic pathway (Jomaa et al. (1999), Science 285, 1573-1576).

Summary of Invention Paragraph (22):
[0022] The invention also relates to substances which are found with the aid of the above described method, with the exception of Fosmidomycin, which is already known to inhibit 1-deoxy-D-xylulose-5-phosphate reductoisomerase (Zeidler et al. (1998), Z. Naturforsch. 53, 980-986).

CLAIMS:

18. Substances which are identified by a method according to claim 17, with the exception of Fosmidomycin.

L4: Entry 11 of 43 File: USPT Oct 16, 2001

DOCUMENT-IDENTIFIER: US 6303365 B1
TITLE: Method of determining activity of 1-deoxy-D-xylulose-5-phosphate reductoisomerase and 1-deoxy-D-xylulose-5-phosphate synthase

Brief Summary Text (6):
This is why the 1-deoxy-xylulose-5-phosphate biosynthetic pathway is of particular interest in the search for new herbicidally active compounds. In particular the two enzymes 1-deoxy-D-xylulose-5-phosphate synthase (DXPS) and 1-deoxy-D xylulose-5-phosphate reductoisomerase (DXPR) are of central importance. It has already been demonstrated that DXPS (CLA I) is essential for the development of a normal plant (Mandel et al. (1996), Plant J. 9, 649-658). This discovery supports the expectation that a herbicidal compound which affects DXPS activity has a herbicidal action. Also, it has been demonstrated that bacterial DXFR is inhibited by the herbicidal compound Fosmidomycin, which is already known (Zeidler et al. (1998), Z. Naturforsch. 53, 980-986; Kuzuyama et al. (1998), Tetrahedron Lett. 39, 7913-7916). However, there are no commercially useable herbicides, which affect DXPS or DXPR activity. In the search for new, improved herbicides, both enzymes are therefore of high importance as sites of action. The 1-deoxy-xylulose-5-phosphate biosynthetic pathway also has importance in microorganisms, especially in parasitic microorganisms as, for example, bacteria or plasmodia. The treatment of infectious diseases, in particular the treatment of malaria, may be based on the inhibition of this metabolic pathway (Jomaa et al. (1999), Science 285, 1573-1576).

Brief Summary Text (22):
The invention also relates to substances which are found with the aid of the above described method, with the exception of Fosmidomycin, which is already known to inhibit 1-deoxy-D-xylulose-5-phosphate reductoisomerase (Zeidler et al. (1998), Z. Naturforsch. 53, 980-986).

L4: Entry 21 of 43 File: USPT Mar 26, 1991

DOCUMENT-IDENTIFIER: US 5002602 A
TITLE: Herbicidal methods and compositions comprising fosmidomycin

Abstract Text (1):
This invention relates to a herbicidal composition comprising fosmidomycin or salt thereof in combination with the other herbicide selected from the group of ametryn or salt thereof or diuron, and to a method of killing weeds by applying to weed seedlings the said combination.

Brief Summary Text (2):
More particularly, it relates to a new herbicidal composition comprising fosmidomycin or salt thereof in combination with the other herbicide selected from the group of ametryn or salt thereof or diuron, and to a method of killing weeds by applying to weed seedlings the said combination.

Brief Summary Text (3):
The fosmidomycin is a known compound, 3-(N-formyl-N-hydroxyamino)propylphosphonic acid as antibacterial agent [Cf. European Journal of Drug Metabobism and Pharmacokinetics Vol. 7, P59 (1982)] and as herbicide [Cf. Japan Kokai No. 106504/1986].

Brief Summary Text (5):
The fundamental physiological action of fosmidomycin resides in the inhibition of production of chlorophyll. Therefore, plants emerging after treatment with fosmidomycin are ready to undergo chlorosis When the treating concentration is such that this chlorosis lasts as long as more than 2 to 3 weeks, arrest of growth occurs as the plant is prevented from nursing itself by photosyntheses, leading to decay. However, as the treating concentration is decreased, the degree and duration of chlorosis are lessened and the plant will not die but show a recovery of growth so that the object of killing cannot be accomplished. While a large variety of herbicides have been developed and put to use for controlling the weeds detrimental to crop plants and the environment, each of these herbicides has its own drawback or shortcoming and none has ever proved fully satisfactory in weed killing effect.

Brief Summary Text (11):
The present inventors discovered that application of a composition containing fosmidomycin or salt thereof in combination with the other herbicide selected from the group of ametryn or salt thereof and diuron to a plant resulted in a surprisingly great synergistic herbicidal effect on the plant. This finding was followed by a further investigation, which culminated in the present invention

Brief Summary Text (12):
The salt of fosmidomycin may include an agronomically acceptable salt thereof such as a base salt (e.g. sodium salt, potassium salt, calcium salt, etc.) and the like.

Brief Summary Text (17):
The ratio of the fosmidomycin or a salt thereof to the ametryn or a salt thereof or diuron in the herbicidal composition of the invention is dependent on the kinds of respective compounds and the kinds of weeds to be controlled.

CLAIMS:

1. A herbicidal composition comprising an herbicidally effective amount of fosmidomycin or a salt thereof in combination with ametryn or a salt thereof, in a ratio of between 10:1 and 1:10, and an arronomically acceptable carrier or carriers.

2. The herbicide composition of claim 1 comprising fosmidomycin or a salt thereof and ametryn.

3. A method of killing broad leaved weeds and grasses, said method comprising applying to broad leaf weeds or grass seedlings a herbicidally effective amount of a combination of fosmidomycin or a salt thereof, and ametryn or a salt thereof, in a ratio of between 1:1 and 1:10.

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☐ 1. Document ID: US 20020119197 A1

L9: Entry 1 of 2 File: PGPB Aug 29, 2002

PGPUB-DOCUMENT-NUMBER: 20020119197
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020119197 A1

TITLE: Process and system for controlled-release drug delivery

PUBLICATION-DATE: August 29, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Dyar, Stephen Craig	Brighton	MI	US	
Fessehaie, Mebrahtu	Mineola	NY	US	
Ghebre-Sellassie, Isaac	Morris Plains	NJ	US	
Mayassi, Monzer Michael	Somerset	NJ	US	
Mollan, Matthew J. JR.	Succasunna	NJ	US	
Woldegaber, Haimonot	Dover	NJ	US	

US-CL-CURRENT: 424/473

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☑ 2. Document ID: US 20020035058 A1

L9: Entry 2 of 2 File: PGPB Mar 21, 2002

PGPUB-DOCUMENT-NUMBER: 20020035058
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020035058 A1

TITLE: Isopentenyl pyrophosphate isomerase (IPI) and/or prenyl transferase inhibitors

PUBLICATION-DATE: March 21, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Brown, Richard John	Sheffield		GB	
Watts, Donald Jeremy	Sheffield		GB	
Russell, Robert Graham Goodwin	Sheffield		GB	
Rogers, Michael John	Slains		GB	

US-CL-CURRENT: 514/1

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L9: Entry 2 of 2 File: PGPB Mar 21, 2002

DOCUMENT-IDENTIFIER: US 20020035058 A1
TITLE: Isopentenyl pyrophosphate isomerase (IPI) and/or prenyl transferase inhibitors

Summary of Invention Paragraph (58):
[0055] For example, the IPI and/or prenyl transferase inhibitors of the present invention find particular utility in the modulation of lipid metabolism, cell proliferation, isoprenoid-related cellular apoptosis and cellular signal transduction. The IPI and/or prenyl transferase inhibitors are also useful as fungicides or herbicides. The inhibitors may have dual inhibitory activity, and inhibit both IPI and prenyl transferase. They may also inhibit other isoprenoid synthetic enzymes.

Summary of Invention Paragraph (100):
[0097] When administered in suitable concentrations, the IPI and/or prenyl transferase inhibitors of the present invention can also be used to block sterol synthesis in plants and fungi. They therefore find utility as fungicides and/or herbicides.

Summary of Invention Paragraph (108):
[0105] Preferably, the IPI and/or prenyl transferase inhibitors have a positively charged nitrogen atom. Particularly preferred are PIBs or IPIBs having a positively charged nitrogen atom. Examples of such bisphosphonates are alendronate, pamidronate and ibandronate, and analogues or derivatives thereof including nitrogen ring-containing (heterocyclic) compounds.

Detail Description Paragraph (6):
[0161] Clodronate, alendronate, ibandronate, YM175 and risedronate were provided by Procter and Gamble Pharmaceuticals, Cincinnati, Ohio. The bisphosphonates were dissolved in PBS, the pH adjusted to 7.4 with 1N NaOH, then filter-sterilised by using a 0.2 .mu.m filter. Mevastatin (also known as compacting was purchased from Sigma Chemical Co, Poole, UK, and converted from the lactone form by dissolving 5 mg mevastatin in 100 .mu.l 1N NaOh. After addition of 1 ml PBS, the pH of the solution was adjusted to approximately pH 8 using 1N HCl, then filter-sterilised. A stock solution of 10 mM mevalonic acid lactone was prepared by dissolving the solid in dry ethanol, while farnesylpyrophosphate (FPP) and geranylgeranylpyrophosphate (GGPP), purchased from Sigma, were dried to remove solvent then resuspended in culture medium immediately before use. Methionine and mevalonolactone was from Amersham, Aylesbury, UK. All other chemicals were from Sigma Chemical Co, Poole, UK, unless stated otherwise.

Detail Description Paragraph (11):
[0166] J774 cells were seeded into 12 well plates (Costar) at a density of 10.sup.5 per well. After 24 h, the medium was replaced with fresh medium containing either 1-100 .mu.M mevastatin, 100 .mu.M alendronate or 15 .mu.M mevastatin, with or without 0.5 .mu.M cycloheximide, 200 .mu.M FPP, 200 .mu.M GGPP or 200 .mu.M mevalonic acid lactone. After 48 h, both adherent and non-adherent cells were collected, fixed with 4% (v/v) formaldehyde than cytospun onto slides and visualised as described by Rogers et al. In addition, DNA was extracted from approximately 5.times.10.sup.5 J774 cells following treatment with 20 .mu.M or 100 .mu.M mevastatin for 48 h, and analysed for the presence of oligonucleosome-sized

fragments by agarose gel electrophoresis.

Detail Description Paragraph (12):
[0167] The effect of cycloheximide, FPP, GGPP and mevalonic acid lactone on loss of total cell viability owing to apoptosis was also assessed by measuring the ability of cells to metabolise MTT (3-(4,5-dimethylthiazol-- 2yl)-2,5-diphenyltetrazolium bromide). 96-well plates (Costar) were inoculated with 1.5.times.10.sup.4 cells per well. 24 h later the cells were treated with 15 .mu.M mevastatin or 100 .mu.M alendronate, together with 0.5 .mu.M cycloheximide, 200 .mu.M FPP, 200 .mu.M GGPP or 200 .mu.M mevalonic acid lactone. After 48 h incubation, the conversation of MTT reagent by viable cells was measured spectrophotometrically according to Rogers et al (1996).

Detail Description Paragraph (16):
[0171] Approximately 10.sup.7 cells in 75 cm.sup.2 flasks were treated for 16 h with 100 .mu.M alendronate or 15 .mu.M mevastatin. Control cells were treated with vehicle (PBS). The cells were then incubated with 8 ml methionine-free medium (containing 100 .mu.M alendronate or 15 .mu.M mevastatin) for 1 h, before addition of 120 .mu.Ci methionine (specific activity 1000 Ci/mmol) and further incubation for 24 h. Adherent and non-adherent cells from each flask were then harvested and lysed in 1.0 ml of RIPA buffer. Lysates for immunoprecipitation of Rab6 were precleared by using 1 .mu.g of rabbit IgG and 20 .mu.l protein A agarose slurry, followed by addition of 2 .mu.g polyclonal rabbit anti-Rab6 (santa Cruz) for 2 h, then 50 .mu.l protein A-agarose and incubation overnight. Ras was immunoprecipitated by overnight incubation, at 4.degree., of 1 ml lysate with 30 .mu.l pan-Ras antibody. Y13-259 conjugated to agarose beads (Oncogene Science). Immunoprecipitates were washed 5 times with 1 ml RIPA buffer, then bound proteins were removed by boiling for 4 minutes in 30 .mu.l Laemmli sample buffer. Finally, samples were electrophoresed on 12.5% polyacrylamide-SDS gels under reducing conditions and detected as described above.

Detail Description Paragraph (19):
[0173] Concentrations of 10-100 .mu.M mevastatin, an inhibitor of HMGCoA reductase (which catalyses the synthesis of mevalonate, FIG. 1) caused a dose-dependent increase in the proportion of J774 cells with the morphological and biochemical features typical of apoptosis i.e. chromatin condensation and formation of apoptotic bodies (FIG. 2) and oligonucleosomal DNA fragmentation (FIG. 3). Mevastatin appeared to be more potent at inducing apoptosis than alendronate or risedronate, since concentrations of approximately 10 .mu.M mevastatin, 30 .mu.M alendronate or 3 .mu.M risedronate caused 50% loss of total cell viability after 48 h (FIG. 4).

Detail Description Paragraph (20):
[0174] Mevastatin-induced loss of cell viability, measured by reduction of MTT reagent, could be prevented by co-incubating J774 cells with 15 .mu.M mevastatin and 0.5 .mu.M cycloheximide during the 48 h culture period (FIG. 5). Analysis of the proportion of morphologically apoptotic cells also demonstrated that cycloheximide prevented apoptosis (not shown). Co-incubation with 200 .mu.M FPP, GGPP or especially mevalonic acid lactone also prevented (at least partially) mevastatin-induced loss of cell viability and apoptosis (FIG. 6). By contrast, apoptosis and loss of cell viability caused by 100 .mu.M alendronate could be partially inhibited only by co-incubation with 200 .mu.M FPP or GGPP but not with 200 .mu.M mevalonic acid lactone (FIG. 6).

Detail Description Paragraph (22):
[0176] J774 cells metabolically-labelled with mevalonolactone for 24 h contained radiolabelled proteins that could be separated by electrophoresis on 12% polyacrylamide gel into proteins of molecular weight 21-26 kDa (mostly geranygeranylated GTP-binding proteins, but also farnesylated Ras proteins), 60-70 kDa (farnesylated lamin B and prelamin A), 17 kDa and 46 kDa. A broad band at the migrating front of the gels (which did not stain with Coomassie blue and was not affected by prior treatment of cell lysates with proteinase K or RNase.sup.14) was most likely radiolabelled, pyrophosphate-containing intermediates of the mevalonate pathway, such as FPP and GGPP. Treatment of J774 cells with 100 .mu.M alendronate, ibandronate and risedronate during the 24 h labelling period markedly reduced the incorporation of radiolabel into all the protein bands (but especially 21-26 kDa,

mostly geranylgeranylated, proteins) and reduced the amount of radiolabelled compounds at the dye front (FIG. 7). 100 .mu.M YM175 inhibited even more effectively the incorporation of radiolabel into proteins and into compounds at the dye front. By contrast, 750 .mu.M clodronate (a concentration that causes a substantial reduction in viability of 3774 cells) did not affect protein prenylation or synthesis of the radiolabelled dye-front compounds. Hence, there was some correlation between the ability of the bisphosphonates to inhibit protein prenylation in J774 cells and the anti-resorptive potency of the bisphosphonates (risedronate>YM175>i bandronate>alendronate>>clodronate). Inhibition of protein prenylation was not the result of an inhibitory effect of the bisphosphonates on de novo protein synthesis, since 24 h treatment with 100 .mu.M of the bisphosphonates does not inhibit incorporation of methionine into protein in J774 cells.

Detail Description Paragraph (24):
[0178] To demonstrate further that both farnesylated and geranylgeranylated proteins were affected by bisphosphonates, we immunoprecipitated Ras and Rab6 proteins from cell lysates of J774 macrophages that had been metabolically labelled with methionine. Immunoprecipitation of Ras from cell lysates of control J774 cells, using the pan-Ras antibody Y13-259, gave rise to two bands of around 21 kD following electrophoresis of immunoprecipitates on 12.5% polyacrylamide gels. These comprised the non-farnesylated form of Ras (the upper band) and farnesylated Ras (the lower band, which migrates faster owing to removal of the terminal tripeptide following prenylation). After treatment of J774 cells for 41 h with 100 .mu.M alendronate (by which time about 80% of the remaining cells were apoptotic) then immunoprecipitation of Ras with the Y13 antibody, the non-farnesylated form was predominant and the farnesylated form became barely detectable (FIG. 8A). Identical results were obtained after treatment with 15 .mu.M mevastatin for 41 h.

Detail Description Paragraph (25):
[0179] Alendronate and mevastatin also prevented geranylgeranylation of Rab6. Immunoprecipitation of Rab6 from lysates of J774 cells that had been treated with 10 .mu.M alendronate or 11 .mu.M mevastatin for 41 h gave rise to a prominent band of 24 kD, non-prenylated Rab6 and a barely detectable 23 kD geranylgeranylated form (FIG. 8B). In lysates from control cells, Rab6 was immunoprecipitated entirely as the geranygeranylated form.

Detail Description Paragraph (27):
[0180] Our observations demonstrate that potent anti-resorptive bisphosphonate drugs such as risedronate, YM175, ibandronate and alendronate can inhibit post-translational modification of proteins with isoprenoid (farnesyl or geranylgeranyl) groups. In 774 macrophages, the incorporation of mevalonate into prenylated proteins, including lamins, Ras and Rab6, was inhibited by a concentration of the bisphosphonates that also causes apoptosis in vitro (100 .mu.M). Furthermore, another inhibitor of protein prenylation, mevastatin, was even more potent than alendronate at causing macrophage apoptosis. Hence, inhibition of protein prenylation is a likely route by which bisphosphonates cause apoptosis in J774 macrophages. The fact that clodronate did not inhibit the incorporation of mevalonate into prenylated proteins, does not inhibit sterol biosynthesis in vitro and is much less potent at causing 3774 apoptosis also supports the view that this bisphosphonate affects cells by a mechanism which is different to that of the more potent bisphosphonates such as risedronate and alendronate.

Detail Description Paragraph (29):
[0182] The exact enzymes of the mevalonate pathway that are inhibited by bisphosphonates remain to be identified. Mevastatin is an inhibitor of HMG-COA reductase and thus prevents synthesis of mevalonate. Hence, mevastatin-induced apoptosis could be prevented by addition of mevalonic acid lactone or the mevalonate-derived compounds FPP and GGPP. By contrast, addition of either FPP or GGPP, but not mevalonic acid lactone, partially prevented alendronate-induced apoptosis. Hence, alendronate appears to inhibit enzymes later in the mevalonate pathway than HMG-CoA reductase. It is possible that both FPP synthase and GGPP synthase are inhibited, since bisphosphonates appeared to prevent the incorporation of mevalonate into FPP and GGPP. However, since FPP is itself converted to GGPP by GGPP synthase, it is also possible that the inhibitory effects of bisphosphonates on both farnesylation and geranylgeranylation of proteins could be the result of

inhibition of FPP synthase alone. Alternatively, BPs could prevent the transfer of prenyl groups to proteins by prenyl protein transferases. It is perhaps more likely that bisphosphonates (like .alpha.-hydroxyfarnesylphosphonate) can actually inhibit several enzymes of the mevalonate pathway that contain similar prenyl-pyrophosphate binding sites. This is supported by the findings of Amin et al, since YM175 and ibandronate (but not alendronate) were potent inhibitors of squalene synthase, whilst we have demonstrated that YM175 and ibandronate probably also inhibit enzymes involved in FPP and GGPP synthesis. The potency of bisphosphonates could therefore depend on the combination of enzymes that are inhibited.

Detail Description Paragraph (31):
[0184] Several studies have suggested that bisphosphonates affect osteoclasts and other cells by interfering with cellular metabolism. Whilst protein tyrosine phosphatases have been postulated to be the molecular targets for alendronate, we have not found any increase in tyrosine phosphorylation in J774 macrophages undergoing apoptosis; inhibition of tyrosine phosphorylation in J774 cells by orthovanadate actually prevents bisphosphonate-induced apoptosis. Furthermore, inhibition of signal transduction processes by direct inhibition of tyrosine phosphatases would be expected to have rapid effects of osteoclasts. However, there are several reports that inhibition of osteoclastic resorption, like induction of osteoclast and J774 apoptosis, occurs many hours after first exposure to bisphosphonates, suggesting that bisphosphonates affect cell metabolism by an indirect effect. Furthermore, our observations that bisphosphonate-induced apoptosis could be partially prevented in the presence of FPP or GGPP clearly suggest that bisphosphonates induce apoptosis as a result of effects on the mavalonate pathway.

Detail Description Table CWU (1):
1 Prenyl transferase IC.sub.50(nM)* IPI Bisphosphonate (FPP synthase) (IPP isomerase) PAMIDRONATE 5000 200 ALENDRONATE 2000 75 YM175 45 35 IBANDRONATE 20 35 RISEDRONATE 20 22 *Concentration required to inhibit enzyme activity by 50%

CLAIMS:

13. A herbicide or fungicide comprising an IPI and/or prenyl transferase inhibitor.

14. Use of an inhibitor of IPI and/or prenyl transferase as a herbicide or fungicide.

22. The use according to any one of the preceding claims wherein the inhibitor is alendronate, pamidronate or ibandronate, or prenyl transferase inhibitory analogues or derivatives thereof.

L9: Entry 1 of 2 File: PGPB Aug 29, 2002

DOCUMENT-IDENTIFIER: US 20020119197 A1
TITLE: Process and system for controlled-release drug delivery

Detail Description Paragraph (2):
[0027] Soluble controlled-release dosage forms of pellets, disks, etc., by which pharmaceutical agents (or active ingredients such as pesticides or herbicides) are controllably released for dissolution to surrounding fluids are well-known. Typically, the pharmaceutically-active agent is disposed in a matrix material and/or coating material which controls the delivery of the pharmaceutically-active agent by controlling either the access of the surrounding bodily fluids to the pharmaceutically-active agent, or controls the release outwardly from the matrix material or coating of the pharmaceutically-active agent.

Detail Description Paragraph (32):
[0057] 29. Other types of active ingredients, that can be formulated according to this invention include acetohexamide, ajamaline, alendronate sodium, amlodipine besylate, amylobarbitone, atorvastatin calcium, simvastatin, pravastatin, fluvastatin, rosuvastatin, bendrofluozide, benzbromarone, benzoate, benzylbenzoate, betametharzone, paroxetine hydrochloride, buproprion hydrochloride, buspirone hydrochloride, chloramphenicol, chloropropamide, chlorthalidone, clofibrate, conjugated estrogens, corticosteroids, diazepam, dicumerol, digitoxin, digoxin, dihydroxypropyltheophylline, diltiazem hydrochloride, doxazosin mesylate, ergot alkaloids, ethotion, felodipine, fluoxetine hydrochloride, fluconazole, fluvastatin sodium, frusemide, glutethimide, griseofulvin, hydrochlorothiazide, hydrocortisone, hydroflumethiazide, hydroquinone, hydroyalkylxanthines, indomethacin, isoxsuprine hydrochloride, ketoprofen, khellin, levothyroxine sodium, losartan potassium, lovastatin, meprobamate, nabilone, nefazodone hydrochloride, nicotinamide, nifedipine, nitrofuirantoin, novalgin, nystatin, papaverine, paracetamol, phenylbutazone, phenobarbitone, pravastin sodium, prednisolone, prednisone, primadonel, reserpine, risperidone, romglizone, salicylic acid, salmeterol xinafoatte, sertraline hydrochloride, simvastatin, spironolactone, sulphabenzamide, sulphadiamadine, sulphamethoxydiazine, sulphamerazine, succinylsulphathiazole, sulphamethizole, sulphamethoxazole, sulphathiazole, sulphisoxazole, sumatriptain succinate, testosterone, tolazoline, tolbutamide, trifluoperazine, trimethoprim, valsartan, zolpidem tartrate and other water insoluble or water soluble active ingredients.

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Search Results - Record(s) 1 through 10 of 20 returned.

☐ 1. Document ID: US 20020058321 A1

L10: Entry 1 of 20 File: PGPB May 16, 2002

PGPUB-DOCUMENT-NUMBER: 20020058321
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020058321 A1

TITLE: Method of determining the activity of 1-deoxy-D-xylulose-5-phosphate reductoisomerase and 1-deoxy-D-xylulose-5-phosphate synthase

PUBLICATION-DATE: May 16, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Martin, William Frank	Neuss	NC	DE	
Hain, Ruediger	Langenfeld		DE	
Tietjen, Klaus-Guenther	Langenfeld		DE	
Busch, Marco	Burscheid		DE	
Kloti, Andreas S.	Durham		US	

US-CL-CURRENT: 435/189; 435/233, 435/320.1, 435/410, 435/69.1, 536/23.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

☐ 2. Document ID: US 6420159 B2

L10: Entry 2 of 20 File: USPT Jul 16, 2002

US-PAT-NO: 6420159
DOCUMENT-IDENTIFIER: US 6420159 B2

TITLE: 1-deoxy-D-xylulose-5-phosphate reductoisomerases, and methods of use

DATE-ISSUED: July 16, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Croteau; Rodney B.	Pullman	WA		
Lange; Bernd M.	Pullman	WA		

US-CL-CURRENT: 435/233

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

☐ 3. Document ID: US 6303365 B1

L10: Entry 3 of 20 File: USPT Oct 16, 2001

US-PAT-NO: 6303365
DOCUMENT-IDENTIFIER: US 6303365 B1

TITLE: Method of determining activity of 1-deoxy-D-xylulose-5-phosphate reductoisomerase and 1-deoxy-D-xylulose-5-phosphate synthase

DATE-ISSUED: October 16, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Martin; William Frank	Neuss			DE
Hain; Ruediger	Langenfeld			DE
Tietjen; Klaus-Guenther	Langenfeld			DE
Busch; Marco	Burscheid			DE
Kloti; Andreas S.	Durham	NC		

US-CL-CURRENT: 435/252.3; 435/320.1, 435/325, 536/23.2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

☐ 4. Document ID: US 6281017 B1

L10: Entry 4 of 20 File: USPT Aug 28, 2001

US-PAT-NO: 6281017
DOCUMENT-IDENTIFIER: US 6281017 B1

TITLE: 1-deoxy-d-xylulose-5-phosphate reductoisomerases and method of use

DATE-ISSUED: August 28, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Croteau; Rodney B.	Pullman	WA		
Lange; Bernd M.	Pullman	WA		

US-CL-CURRENT: 435/468; 435/189, 435/233, 435/320.1, 435/410, 435/476

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

☐ 5. Document ID: US 6207178 B1

L10: Entry 5 of 20 File: USPT Mar 27, 2001

US-PAT-NO: 6207178

DOCUMENT-IDENTIFIER: US 6207178 B1

TITLE: Solid lipid particles, particles of bioactive agents and methods for the manufacture and use thereof

DATE-ISSUED: March 27, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Westesen; Kirsten	Konigslutter/Bornum			DE
Siekmann; Britta	Braunschweig			DE

US-CL-CURRENT: 424/405; 252/363.5, 264/4.4, 424/400, 424/497, 424/498, 504/362, 514/937, 514/964, 516/77, 516/926, 516/928

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 6. Document ID: US 6197349 B1

L10: Entry 6 of 20 File: USPT Mar 6, 2001

US-PAT-NO: 6197349
DOCUMENT-IDENTIFIER: US 6197349 B1

TITLE: Particles with modified physicochemical properties, their preparation and uses

DATE-ISSUED: March 6, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Westesen; Kirsten	Konigslutter			DE
Siekmann; Britta	Sodertalje			SE

US-CL-CURRENT: 424/501; 264/4.1, 264/4.3, 264/4.33, 264/4.4, 424/502, 427/213.36, 428/402.21, 514/772.3

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 7. Document ID: US 5885486 A

L10: Entry 7 of 20 File: USPT Mar 23, 1999

US-PAT-NO: 5885486
DOCUMENT-IDENTIFIER: US 5885486 A

TITLE: Solid lipid particles, particles of bioactive agents and methods for the manufacture and use thereof

DATE-ISSUED: March 23, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Westesen; Kirsten	Konigslutter/Bornum			DE
Siekmann; Britta	Braunschweig			DE

US-CL-CURRENT: 428/402.24; 516/40, 516/77, 516/926

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 8. Document ID: US 5785976 A

L10: Entry 8 of 20 File: USPT Jul 28, 1998

US-PAT-NO: 5785976
DOCUMENT-IDENTIFIER: US 5785976 A

TITLE: Solid lipid particles, particles of bioactive agents and methods for the manufacture and use thereof

DATE-ISSUED: July 28, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Westesen; Kirsten	Konigslutter/Bornum			DE
Siekmann; Britta	Braunschweig			DE

US-CL-CURRENT: 424/400; 252/363.5, 264/4.4, 424/405, 424/484, 424/498, 427/213.32, 428/402.24, 504/363, 514/965, 516/31 , 516/77, 516/9, 516/922, 516/926, 516/930

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 9. Document ID: US 5002602 A

L10: Entry 9 of 20 File: USPT Mar 26, 1991

US-PAT-NO: 5002602
DOCUMENT-IDENTIFIER: US 5002602 A

TITLE: Herbicidal methods and compositions comprising fosmidomycin

DATE-ISSUED: March 26, 1991

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Kamuro; Yasuo	Ibaraki			JP
Kawai; Tadahide	Ibaraki			JP
Kakiuchi; Toshihito	Ibaraki			JP

US-CL-CURRENT: 504/128

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

☑ 10. Document ID: US 4846872 A

L10: Entry 10 of 20 File: USPT Jul 11, 1989

US-PAT-NO: 4846872
DOCUMENT-IDENTIFIER: US 4846872 A

TITLE: Herbicide

DATE-ISSUED: July 11, 1989

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Kamuro; Yasuo	Ibaraki			JP
Kawai; Tadahide	Ibaraki			JP
Kakiuchi; Toshihito	Ibaraki			JP

US-CL-CURRENT: 504/127; 504/128

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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Terms	Documents
L6 and l8	20

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☑ 11. Document ID: JP 63152306 A

L10: Entry 11 of 20 File: JPAB Jun 24, 1988

PUB-NO: JP363152306A
DOCUMENT-IDENTIFIER: JP 63152306 A
TITLE: HERBICIDE

PUBN-DATE: June 24, 1988

INVENTOR-INFORMATION:

NAME	COUNTRY
KAMURO, YASUO	
KAWAI, TADAHIDE	
KAKIUCHI, TOSHIHITO	

INT-CL (IPC): A01N 57/20; A01N 63/02

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☑ 12. Document ID: JP 61106504 A

L10: Entry 12 of 20 File: JPAB May 24, 1986

PUB-NO: JP361106504A
DOCUMENT-IDENTIFIER: JP 61106504 A
TITLE: HERBICIDE

PUBN-DATE: May 24, 1986

INVENTOR-INFORMATION:

NAME	COUNTRY
YAMAJI, TEIZO	
AZUMA, SHIZUO	
HIRAMATSU, TOSHIYUKI	
ICHIKAWA, YATARO	

INT-CL (IPC): A01N 57/02

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 13. Document ID: DE 19935967 A1

L10: Entry 13 of 20 File: EPAB Feb 1, 2001

PUB-NO: DE019935967A1
DOCUMENT-IDENTIFIER: DE 19935967 A1
TITLE: New DNA encoding Arabidopsis thaliana 1-deoxy-D-xylulose-5-phosphate reductoisomerase for identifying modulators of the enzyme that can be used as herbicides, antibiotic or anti-malarial agents

PUBN-DATE: February 1, 2001

INVENTOR-INFORMATION:

NAME	COUNTRY
HAIN, RUEDIGER	DE
TIETJEN, KLAUS-GUENTHER	DE
BUSCH, MARCO	DE
MARTIN, WILLIAM F PROF DR	DE

INT-CL (IPC): C07 H 21/00; C12 N 15/60; C12 N 15/11; C12 N 15/12; C12 N 5/10; C12 Q 1/00; A01 N 63/00; A01 N 37/44
EUR-CL (EPC): C12N009/04; C12N009/10, C12Q001/32 , C12Q001/48 , C12Q001/26 , C12N009/90

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 14. Document ID: AU 200215352 A WO 200231120 A1

L10: Entry 14 of 20 File: DWPI Apr 22, 2002

DERWENT-ACC-NO: 2002-444176
DERWENT-WEEK: 200254
COPYRIGHT 2002 DERWENT INFORMATION LTD
TITLE: Novel transgenic host cell useful for screening antibacterial and herbicidal agents, comprising a disruption of endogenous gene in methylerythritol phosphate pathway, and transgene that replaces disrupted gene

INVENTOR: CORNISH, R; HAHN, F ; POULTER, C ; TESTA, C

PRIORITY-DATA: 2000US-240253P (October 13, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
AU 200215352 A	April 22, 2002		000	C12N001/20
WO 200231120 A1	April 18, 2002	E	036	C12N001/20

INT-CL (IPC): A01 N 43/04; A61 K 31/70; A61 K 38/00; C12 N 1/20; C12 Q 1/18

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 15. Document ID: EP 1204756 A2 WO 200109341 A2 DE 19935967 A1 AU 200068269 A US 6303365 B1 US 20020058321 A1

L10: Entry 15 of 20 File: DWPI May 15, 2002

DERWENT-ACC-NO: 2001-159877
DERWENT-WEEK: 200239

TITLE: New DNA encoding Arabidopsis thaliana 1-deoxy-D-xylulose-5-phosphate reductoisomerase for identifying modulators of the enzyme that can be used as herbicides, antibiotic or anti-malarial agents

INVENTOR: BUSCH, M; HAIN, R ; MARTIN, W F ; TIETJEN, K ; KLOETI, A S ; KLOTI, A S

PRIORITY-DATA: 1999US-0449335 (November 24, 1999), 1999DE-1035967 (July 30, 1999)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
EP 1204756 A2	May 15, 2002	E	000	C12N015/53
WO 200109341 A2	February 8, 2001	E	029	C12N015/53
DE 19935967 A1	February 1, 2001		000	C07H021/00
AU 200068269 A	February 19, 2001		000	C12N015/53
US 6303365 B1	October 16, 2001		000	C12N001/20
US 20020058321 A1	May 16, 2002		000	C12N009/02

INT-CL (IPC): A01 N 37/44; A01 N 63/00; C07 H 21/00; C07 H 21/04; C12 N 1/20; C12 N 1/21; C12 N 5/04; C12 N 5/10; C12 N 9/02; C12 N 9/04; C12 N 9/10; C12 N 9/90; C12 N 15/00; C12 N 15/11; C12 N 15/12; C12 N 15/53; C12 N 15/54; C12 N 15/60; C12 P 21/02; C12 Q 1/00; C12 Q 1/32; C12 Q 1/48

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 16. Document ID: WO 200017212 A1 AU 9963287 A

L10: Entry 16 of 20 File: DWPI Mar 30, 2000

DERWENT-ACC-NO: 2000-303195
DERWENT-WEEK: 200103

TITLE: New fosmidomycin derivatives useful in medicine for control of viral, bacterial, fungal and parasiticidal infections and in plant protection as fungicides, bactericides and herbicides

INVENTOR: JOMAA, H

PRIORITY-DATA: 1998DE-1043223 (September 22, 1998)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 200017212 A1	March 30, 2000	G	033	C07F009/40
AU 9963287 A	April 10, 2000		000	C07F009/40

INT-CL (IPC): A01 N 57/18; A61 K 31/66; C07 F 9/40

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 17. Document ID: BR 9913990 A WO 200016757 A2 AU 9959811 A NO 200101430 A EP 1115388 A1 CZ 200100989 A3 SK 200100393 A3

L10: Entry 17 of 20 File: DWPI Oct 23, 2001

DERWENT-ACC-NO: 2000-283424
DERWENT-WEEK: 200172

TITLE: Use of fosmidomycin derivatives in medicine for control of parasiticidal, fungal, viral and bacterial infections and in plant protection as fungicides, bactericides and herbicides

INVENTOR: JOMAA, H

PRIORITY-DATA: 1998DE-1043222 (September 22, 1998)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
BR 9913990 A	October 23, 2001		000	A61K031/00
WO 200016757 A2	March 30, 2000	G	024	A61K031/00
AU 9959811 A	April 10, 2000		000	A61K031/00
NO 200101430 A	May 9, 2001		000	A61K031/00
EP 1115388 A1	July 18, 2001	G	000	A61K031/00
CZ 200100989 A3	July 11, 2001		000	A61K031/66
SK 200100393 A3	August 6, 2001		000	A61K031/00

INT-CL (IPC): A01 N 57/22; A61 K 31/00; A61 K 31/66; A61 P 31/04; A61 P 31/10; A61 P 31/14; A61 P 33/00

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw. Desc | Clip Img | Image

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☐ 18. Document ID: CN 1348374 A WO 9952938 A2 DE 19828097 A1 DE 19831637 A1 AU 9944816 A DE 19923567 A1 DE 19831639 C1 BR 9909669 A CZ 200003500 A3 EP 1071959 A2 CN 1297532 A SK 200001523 A3 KR 2001034783 A HU 200101711 A2 KR 2001042692 A KR 2001070962 A KR 2001075259 A CN 1319134 A MX 2001000488 A1 JP 2002511486 W

L10: Entry 18 of 20 File: DWPI May 8, 2002

DERWENT-ACC-NO: 1999-611286
DERWENT-WEEK: 200253

TITLE: Identifying antiparasitic agents used to treat or prevent parasitic infections, especially malaria, sleeping sickness and leishmaniosis

INVENTOR: HASSAN, J; JOMAA, H

PRIORITY-DATA: 1998DE-1043279 (September 22, 1998), 1998DE-1016196 (April 14, 1998), 1998DE-1025585 (June 9, 1998), 1998DE-1028097 (June 24, 1998), 1998DE-1031637 (July 15, 1998), 1998DE-1031638 (July 15, 1998), 1998DE-1031639 (July 15, 1998), 1998DE-1043222 (September 22, 1998), 1998DE-1043223 (September 22, 1998), 1998DE-1043360 (September 22, 1998)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
CN 1348374 A	May 8, 2002		000	A61K031/66
WO 9952938 A2	October 21, 1999	G	068	C07K014/00
DE 19828097 A1	December 30, 1999		000	A61K031/66
DE 19831637 A1	January 27, 2000		000	A61K031/66
AU 9944816 A	November 1, 1999		000	
DE 19923567 A1	April 6, 2000		000	C07K014/445
DE 19831639 C1	May 11, 2000		000	C07F009/38
BR 9909669 A	December 19, 2000		000	C07K014/00
CZ 200003500 A3	January 17, 2001		000	C07K014/435
EP 1071959 A2	January 31, 2001	G	000	G01N033/68
CN 1297532 A	May 30, 2001		000	G01N033/68
SK 200001523 A3	May 10, 2001		000	C07K014/00
KR 2001034783 A	April 25, 2001		000	A61K031/66
HU 200101711 A2	September 28, 2001		000	C07K014/00
KR 2001042692 A	May 25, 2001		000	C07K014/00
KR 2001070962 A	July 28, 2001		000	C07F009/38
KR 2001075259 A	August 9, 2001		000	C12N015/61
CN 1319134 A	October 24, 2001		000	C12N009/90
MX 2001000488 A1	May 1, 2001		000	A01N057/18
JP 2002511486 W	April 16, 2002		073	C07K014/00

A , KR 2001070962 A , KR 2001075259 A INT-CL (IPC): A01 H 5/00; A01 N 57/18; A01 N 57/20; A61 K 31/66; A61 K 31/662; A61 K 31/663; A61 K 38/55; A61 K 45/00; A61 K 45/06; A61 P 33/00; A61 P 33/02; A61 P 33/06; A61 P 33/10; A61 P 43/00; C07 F 9/38; C07 F 9/46; C07 K 14/00; C07 K 14/435; C07 K 14/445; C07 K 16/00; C07 K 16/18; C12 N 5/10; C12 N 9/10; C12 N 9/12; C12 N 9/88; C12 N 9/90; C12 N 15/09; C12 N 15/52; C12 N 15/61; C12 N 15/79; C12 P 21/02; C12 Q 1/48; C12 Q 1/527; C12 Q 1/68; G01 N 33/68; A61 K 31/662; A61 K 31:366

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☑ 19. Document ID: JP 63152306 A

L10: Entry 19 of 20 File: DWPI Jun 24, 1988

DERWENT-ACC-NO: 1988-216575
DERWENT-WEEK: 198831

TITLE: New herbicide with synergistic weeding action - comprises fosmidomycin or its salt, and ametryn or its salt, or diuron

PRIORITY-DATA: 1986JP-0188086 (August 11, 1986), 1987JP-0200695 (August 10, 1987)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 63152306 A	June 24, 1988		006	

INT-CL (IPC): A01N 43/70; A01N 47/30; A01N 57/20; A01N 63/02

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 20. Document ID: EP 256785 A DE 3767075 G EP 256785 B US 4846872 A US 5002602 A

L10: Entry 20 of 20 File: DWPI Feb 24, 1988

DERWENT-ACC-NO: 1988-051532
DERWENT-WEEK: 198808

TITLE: Herbicide contg. fosmidomycin and ametryn or diuron - provides synergistic pre=emergence control over broadleaf weeds and grasses

INVENTOR: KAKIUCHI, T; KAMURO, Y ; KAWAI, T

PRIORITY-DATA: 1986JP-0188085 (August 11, 1986)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
EP 256785 A	February 24, 1988	E	014	
DE 3767075 G	February 7, 1991		000	
EP 256785 B	December 27, 1990		000	
US 4846872 A	July 11, 1989		004	
US 5002602 A	March 26, 1991		000	

INT-CL (IPC): A01N 43/70; A01N 47/30; A01N 57/20

L10: Entry 10 of 20 File: USPT Jul 11, 1989

DOCUMENT-IDENTIFIER: US 4846872 A
TITLE: Herbicide

Abstract Text (1):
This invention relates to a herbicidal composition comprising fosmidomycin or salt thereof in combination with the other herbicide selected from the group of ametryn or salt thereof or diuron, and to a method of killing weeds by applying to weed seedlings the said combination.

Brief Summary Text (1):
This invention relates to a new herbicide.

Brief Summary Text (2):
More particularly, it relates to a new herbicidal composition comprising fosmidomycin or salt thereof in combination with the other herbicide selected from the group or ametryn or salt thereof or diuron, and to a method of killing weeds by applying to weed seedlings the said combination.

Brief Summary Text (3):
The fosmidomycin is a known compound, 3-(N-formyl-N-hydroxyamino)propylphosphonic acid as antibacterial agent [Cf. European Journal of Drug Metabolism and Pharmacokinetics Vol. 7, P59 (1982)] and as herbicide [Cf. Japan Kokai No. 106504/1986].

Brief Summary Text (4):
Further, ametryn and diuron are also known herbicides, 2-methylmercapto-4-ethylamino-6-isopropylamino-s-triazine and 3-(3,4-Dichlorophenyl)-1,1-dimethylurea, respectively [Cf. The Merck Index tenth edition items 392 and 3400 (1983)].

Brief Summary Text (5):
The fundamental physiological action of fosmidomycin resides in the inhibition of production of chlorophyll. Therefore, plants emerging after treatment with fosmidomycin are ready to undergo chlorosis. When the treating concentration is such that this chlorosis lasts as long as more than 2 to 3 weeks, arrest of growth occurs as the plant is prevented from nursing itself by photosyntheses, leading to decay. However, as the treating concentration is decreased, the degree and duration of chlorosis are lessened and the plant will not die but show a recovery of growth so that the object of killing cannot be accomplished. While a large variety of herbicides have been developed and put to use for controlling the weeds detrimental to crop plants and the environment, each of these herbicides has its own drawback or shortcoming and none has ever proved fully satisfactory in weed killing effect.

Brief Summary Text (6):
Thus, what are mainly desired in herbicides are:

Brief Summary Text (7):
(1) The maximum possible coverage of weed varieties that can be controlled (a broad herbicidal spectrum)

Brief Summary Text (10):
(4) Reduced amounts of active substances required for control (reduced herbicide

consumption)

Brief Summary Text (11):
The present inventors discovered that application of a composition containing fosmidomycin or salt thereof in combination with the other herbicide selected from the group of ametryn or salt thereof and diuron to a plant resulted in a surprisingly great synergistic herbicidal effect on the plant. This finding was followed by a further investigation, which culminated in the present invention.

Brief Summary Text (12):
The salt of fosmidomycin may include an agronomically acceptable salt thereof such as a base salt (e.g. sodium salt, potassium salt, calcium salt, etc.) and the like.

Brief Summary Text (14):
The herbicidal composition according to the present invention displays remarkable efficacy as a postemergence herbicide, and is preferably applied to the whole stalks and foliage of weeds that have emerged.

Brief Summary Text (15):
Moreover, the herbicidal composition of the present invention provides effective control, irrespective of weed variety, e.g. broad-leaved weeds and grasses.

Brief Summary Text (16):
The application rate for the active ingredients in the herbicidal composition of the invention varies according to the combination used and kinds of weeds to be controlled. Generally however, the optimum rate of application is selected from the range of 5 to 1000 grams/10 ares and preferably from the range of 100 to 500 grams/10 ares.

Brief Summary Text (17):
The ratio of the fosmidomycin or a salt thereof to the ametryn or a salt thereof or diuron in the herbicidal composition of the invention is dependent on the kinds of respective compounds and the kinds of weeds to be controlled.

Brief Summary Text (19):
To apply the herbicidal composition of the invention, it can be mixed with a carrier suited to the intended usage and applied in such varied forms as dusts, granular preparations, wettable powders, liquid preparations, emulsifiable concentrates, flowable emulsion concentrates and so on. The carrier mentioned just above may be a solid or a liquid carrier or a combination thereof. As examples of said carrier, there may be mentioned finely divided minerals such as kaolinite, bentonite, pyrophyllite, talc, diatomaceous earth, silica gel, calcium carbonate, etc., finely divided vegetable materials such as starch, gum arabic, etc., organic solvents such as alcohols, ketones, kerosine, benzene, toluene, xylene, cyclohexane, methylnaphthalene, dioxane, dimethylformamide, dimethyl sulfoxide, corn oil, o-dichlorobenzene, isophorone, water and so on. Further, agronomically acceptable adjuvants and auxiliaries such as wetting agents, dispersing agents, adhesives, extenders, etc. can be incorporated, if necessary, in appropriate proportions.

Brief Summary Text (20):
Each of these preparations is not only useful as such but may be used in combination with bactericides, fungicides, nematocides, insecticides, plant growth regulators, fertilizers, other herbicides and so on.

Detailed Description Text (18):
Two weeks after the treatment, herbicidal effect of the test compounds on the weeds were observed and scored on a rating scale of 0 for no effect through--100 for complete kill.

Detailed Description Text (26):
As clear from the data listed in the above table (a) and (b), all combinations of the test compound A and B can be judged to have synergistic herbicidal activity.

Detailed Description Text (31):
As clear from the data listed in the above table (a) and (b), all combinations of

the test compound A and C can be judged to have synergistic herbicidal activity.

Detailed Description Paragraph Table (3):
Com- Time pound after No. appli- Plant No. (concen- cation in Herbicidal activity (%) tration days 1 2 3 4 5 6 7
1 10 .circle.22 .circle.27 .circle.34 .circle.40 .circle.46 .circle.25 .circle.28 (500 ppm) 20 .circle.56 .circle.44 .circle.61 .circle.67 .circle.60 .circle.40 .circle.43 30 38 31 55 52 42 30 27 1 10 .circle.51 .circle.65 .circle.72 .circle.78 .circle.59 .circle.52 .circle.69 (5000 20 100 100 100 100 100 100 100 ppm) 30 100 100 100 100 100 100 100

Detailed Description Paragraph Table (4):
Compound No. Chemical name (concentration)
1
2-Methylmercapto-4-ethylamino-6-isopropylamino s-triazine 1* (500 ppm) 2
2-Methylmercapto-4-ethylamino-6-isopropylamino s-triazine 1* (500 ppm) +
3-(N--Formyl-N--hydroxyamino)propylphosphonic acid monosodium salt (500 ppm) 3
3-(N--Formyl-N--hydroxyamino)propylphosphonic acid monosodium salt (500 ppm)
Note As test compounds 1*, the corresponding commercial product Gesapax (25% emulsifiable concentrate, CibaGeigy) was used.

Detailed Description Paragraph Table (7):
Test compound and application rate (g/10a) Test weed and herbicidal activity (%) B A a b c d e f 0 25 5 5 5 10 10 0 0 50 10 10 10 15 10 10 0 100 20 20 20 30 15 10 62.5 0 0 0 0 0 0 0 125 0 20 20 15 20 10 15 250 0 40 50 60 60 60 55 62.5 25 60 60 50 60 70 60 125 25 70 65 70 80 70 70 250 25 85 80 85 80 85 80 62.5 50 70 70 75 80 70 70 125 50 80 80 90 90 80 80 250 50 90 >90 90 >90 90 90 62.5 100 80 90 80 >90 80 80 125 100 90 >90 >90 >90 90 90 250 100 >90 >90 >90 >90 >90 >90 C A a b c d e f
0 25 5 5 5 10 10 0 0 50 10 10 10 25 10 10 0 100 20 20 20 30 15 10 250 0 5 5 10 5 10 5 500 0 15 10 20 15 20 15 750 0 40 30 40 30 40 30 250 25 40 50 30 50 50 40 500 25 60 60 40 50 55 60 750 25 85 80 75 85 85 85 250 50 60 70 50 70 60 60 500 50 80 90 80 90 90 85 750 50 90 90 90 >90 90 90 250 100 85 90 80 90 80 70 500 100 90 >90 >90 >90 >90 >90 750 100 >90 >90 >90 >90 >90 >90

Detailed Description Paragraph Table (8):
application rate (g/10a) of the test compound A 0 25 50 100 application rate (g/10a) 0 100 95 85 85 of the test compound B 62.5 100 45 25 10 125 80 20 10 <10 250 50 10 10 <10
Note: Test compound A: 3(N--formyl-N--hydroxyamino)-propylphosphonic acid monosodium salt Test compound B: 2methylmercapto-4-ethylamino-6-isopropylamino-s-triazine [Gesapax (25% emulsifiable concentrate, CibaGeigy)]-

Other Reference Publication (1):
Yamaji et al., "N-substituted alkyl amine phosphates as herbicides" CA 105: 166897j (1986).

CLAIMS:

1. A herbicidal composition comprising a herbicidally effective amount of fosmidomycin or a salt thereof in combination with the herbicide diuron, in a ratio of between 10:1 and 1:10, and an agronomically acceptable carrier or carriers.

2. A method of killing broad leaved weeds and grasses, said method comprising applying to broad leaf weeds or grass seedlings a herbicidally effective amount of a combination of fosmidomycin or a salt thereof and the herbicide diuron.

3. The herbicidal composition of claim 1 wherein said ratio is between 6:1 and 1:6.

L10: Entry 11 of 20 File: JPAB Jun 24, 1988

DOCUMENT-IDENTIFIER: JP 63152306 A
TITLE: HERBICIDE

Abstract (1):
PURPOSE: To obtain a herbicide having synergistically raised herbicidal action and showing excellent effects as a herbicide after germination of weeds, by using both fosmidomycin or a salt thereof and another herbicide selected from ametryn or a salt thereof and diuron.

Abstract (2):
CONSTITUTION: A herbicide showing synergistically herbicidal actions which can not be expected by simple application of each compound, by blending (A) fosmidomycin [3-(N-formyl-N-hydroxyamino)propylphosphonic acid] with (B) a compound selected from ametryn (2-methylmercapto-4-ethylamino-6- isopropylamino-s-triazine) or a salt thereof and diuron [3-(3,4-dichlorophenyl)-1,1- dimethylurea] as essential components in a blending ratio of the component A:B=10:1∼1:10, preferably 6:1∼1:6 and applying the blend to plants. Treatment of the whole stems and leave of germinated weeds is proper as the treating method.

L10: Entry 12 of 20 File: JPAB May 24, 1986

DOCUMENT-IDENTIFIER: JP 61106504 A
TITLE: HERBICIDE

Abstract (1):
PURPOSE: To provide a herbicide containing a specific compound as a main active component, capable of killing a wide variety of weeds such as annual weeds and perennial weeds or suppressing the growth of the weeds by chlorosis, exhibiting excellent herbicidal activity and useful for the control of weeds in uncultivated land.

Abstract (2):
CONSTITUTION: The objective herbicide contains the N-substituted alkylamine phosphoric acid derivative of formula I (R1 is H, OH, lower alkyl or phenyl; R2 and R4 are H or lower alkyl; R3 is H or formyl; n is 1∼5), e.g. 3-(N-formyl-N-hydroxyamino)propylphosphonic acid isopropylamine salt, etc. as an active component. The compound of formula I wherein R1 is OH, R2 and R4 are H, R3 is formyl and n is 3 can be prepared e.g. by reacting the compound of formula II with the compound of formula III by Michaelis-Aransor reaction, reacting the resultant compound of formula IV with the compound of formula V to obtain the compound of formula VI, decomposing the compound with an acid, and finally reacting with formic acid in acetic anhydride to effect the N- formylation.

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L10: Entry 16 of 20 File: DWPI Mar 30, 2000

DERWENT-ACC-NO: 2000-303195
DERWENT-WEEK: 200103

TITLE: New fosmidomycin derivatives useful in medicine for control of viral, bacterial, fungal and parasiticidal infections and in plant protection as fungicides, bactericides and herbicides

INVENTOR: JOMAA, H

PATENT-ASSIGNEE: JOMAA H (JOMAI)

PRIORITY-DATA: 1998DE-1043223 (September 22, 1998)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 200017212 A1	March 30, 2000	G	033	C07F009/40
AU 9963287 A	April 10, 2000		000	C07F009/40

DESIGNATED-STATES: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DK DM EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW

APPLICATION-DATA:

PUB-NO	APPL-DATE	APPL-NO	DESCRIPTOR
WO 200017212A1	September 22, 1999	1999WO-EP07053	
AU 9963287A	September 22, 1999	1999AU-0063287	
AU 9963287A		WO 200017212	Based on

INT-CL (IPC): A01 N 57/18; A61 K 31/66; C07 F 9/40

RELATED-ACC-NO: 2000-283424;2000-283543 ;2001-025196

ABSTRACTED-PUB-NO: WO 200017212A
BASIC-ABSTRACT:

NOVELTY - Aminoalkyl and aminoalkenyl substituted phosphinoyl, phosphinic acid and phosphonic acid derivatives are new.

DETAILED DESCRIPTION - The derivatives are compounds (I) and their salts, esters and ester salts:

R1 and R2 = H; halo; OX1; OX2; or alkyl, hydroxyalkyl, alkenyl, alkynyl, aryl, acyl, cycloalkyl, aralkyl or heterocyclyl (each optionally substituted);

X1 and X2 = H; or alkyl, hydroxyalkyl, alkenyl, alkynyl, aryl, acyl, cycloalkyl, aralkyl or heterocyclyl (each optionally substituted);

A = alkylene; hydroxyalkylene; or alkenylene;

R3 = halo; OX3; or alkyl, hydroxyalkyl, alkenyl or alkynyl with up to 26C, aryl, acyl, aralkyl, cycloalkyl or heterocyclyl (each optionally substituted);

X3 = H; alkyl, hydroxyalkyl, alkenyl or alkynyl with up to 26C, aryl, aralkyl, cycloalkyl or heterocyclyl (each optionally substituted); silyl;

organic or inorganic cation, especially a group Ia, IIa or IIIa metal, optionally substituted ammonium or group derived from ethylenediamine or an amino acid;

R4 = alkyl, hydroxyalkyl, alkenyl or alkynyl with 10-26C (each optionally substituted); or OX4;

X4 = alkyl, hydroxyalkyl, alkenyl or alkynyl with 10-26C (each optionally substituted).

An INDEPENDENT CLAIM is also included for a pharmaceutical preparation containing a compound (I) and optionally:

(a) sulfadoxine, artemisinine, atovaquone, quinine, chloroquine, hydroxychloroquine, mefloquine, halofantrine, pyrimethamine, armensine, tetracycline, doxycycline, proguanil, metronidazole, praziquantil, niclosamide, mebendazole, pyrantel, tiabendazole, diethylcarbazine, piperazine, pyrivinium, metrifonate, oxamniquine, bithional or suramin; or

(b) penicillin, benzylpenicillin, phenoxypenicillin, isoxazolylpenicillin, aminopenicillin, ampicillin, amoxicillin, bacampicillin, carboxypenicillin, ticarcillin, temocillin, acylaminopenicillins, azlocillin, mezlocillin, piperacillin, apalcillin, mecillinam, cefoxitin, cefotetan, cefmetazole, latamofex, flomoxef, cefozidime, ceftazidime, cefpirome, cefepime, cefsulodin, cefoperazone, loracarbef, cefprozil, cefixime, cefpodoxime proxetil, cefuroxime axetil, cefetamet, cefotiam hexetil, cefdinir, cefibuten, carbapenem, imipenem/cilastatin, biapenem, aztreonam or (in description only) clavulanic acid/amoxycillin, clavulanic acid/ticarcillin, sulbactam/ampicillin, tazobactam/piperacillin, oxytetracycline, rolitetracycline, doxycycline, minocycline, chloramphenicol, gentamycin, tobramycin, netilmycin, amikacin, spectionomyxin, erythromycin, clarithromycin, roxithromycin, azithromycin, dirithromycin, spiramycin, josamycin, lincosamide, clindamycin, fusidic acid, vancomycin, tecoplanin, fosfomycin, co-trimoxazole, trimethoprim, nitrofurantoin, nitrofurazone, norfloxacin, ciprofloxacin, ofloxacin, sparfloxacin, enoxacin, fleroxacin, pefloxacin, lomefloxacin, Bay Y3118, isoniazid, rifampicin, rifabutin, ethambutol, pyrazinamide, streptomycin, capreomycin, prothionamide, terizidone, dapsone, clofazimine, bacitracin, tyrothricin, neomycin, kanamycin, paromomycin, mupirocin, acyclovir, ganciclovir, azidothymidine, didanosine, zalcitabin, thiacytidine, stavudin, ribavirin, idoxuridine, trifluridine, foscarnet, amantadine , interferons, amphotericin B, nystatin, natamycin, miconazole, ketoconazole, itraconazole, fluconazole, UK-109,496, clotrimazole, econazole, isoconazole, oxiconazole, bifonazole, flucytosine, griseofulvin, ciclopiroxolamine, tolnaftate, naftifine, terbinafine, amorolfine, betulinic acid, quinine,quinidine, mefloquine, halofantrine, chloroquine, amodiaquine, acridine, benzonaphthyridine, mepacrine, pyronaridine, sulfonamides, sulfadoxine, sulfalene, trimethoprim, proguanil, diaminopyrimidines, pyrimethamine, primaquine, aminoquinolines, WR 238,605, dihydroartemisinin, 10b artemether, arteether, artesunate, atovaquone, suramin, melarsoprol, nifurtmox, stibogluconate sodium, pentamidine, clioquinol, ivermectin or embonate.

ACTIVITY - Virucidal; bactericidal; fungicidal, parasiticidal; herbicidal.

MECHANISM OF ACTION - None given.

USE - Compounds (I) are useful in human and veterinary medicine for the prevention and treatment of infections caused by viruses, bacteria, fungi and parasites. They are also useful in plant protection as fungicides, bactericides and herbicides.

ADVANTAGE - Compounds (I) have less side effects than known drugs and plant protection agents.

ABSTRACTED-PUB-NO: WO 200017212A
EQUIVALENT-ABSTRACTS:

CHOSEN-DRAWING: Dwg.0/0

DERWENT-CLASS: B05 C01
CPI-CODES: B05-B01B; B05-B01E; B05-B01F; B05-B01G; B14-A01; B14-A02; B14-A03; C05-B01B; C05-B01E; C05-B01F; C05-B01G; C14-A01; C14-A02; C14-A03;

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L10: Entry 18 of 20 File: DWPI May 8, 2002

DERWENT-ACC-NO: 1999-611286
DERWENT-WEEK: 200253

TITLE: Identifying antiparasitic agents used to treat or prevent parasitic infections, especially malaria, sleeping sickness and leishmaniosis

Basic Abstract Text (10):
(h) (A), or herbicides, identified using (I).

Basic Abstract Text (11):
ACTIVITY - Antimalarial; antiparasitic; antibacterial; antifungal; antiviral; herbicide.

Basic Abstract Text (13):
USE - (A) are used (i) to treat or prevent parasitic infections, especially malaria, sleeping sickness and leishmaniosis, but also those caused by fungi, bacteria and viruses, and are useful in human or veterinary medicine, and (ii) as herbicides. Mice were given an intraperitoneal dose of 50 mg/kg 3-(N-formyl-N-hydroxyamino)propylphospho- nic acid monosodium salt (an identified (A)), then the next day inoculated with P. vinckeii. No infection was detected in treated animals, which remained parasite-free even after 8 weeks, although untreated animals had over 80% parasitemia after 5 days.

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☑	JP61106504A	all	all	N/A	USPT,PGPB,JPAB,EPAB,DWPI
☐	JP363152306A	all	all	6	USPT,PGPB,JPAB,EPAB,DWPI

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L10: Entry 19 of 20 File: DWPI Jun 24, 1988

DERWENT-ACC-NO: 1988-216575
DERWENT-WEEK: 198831

TITLE: New herbicide with synergistic weeding action - comprises fosmidomycin or its salt, and ametryn or its salt, or diuron

PATENT-ASSIGNEE: FUJISAWA PHARM CO LTD (FUJI)

PRIORITY-DATA: 1986JP-0188086 (August 11, 1986), 1987JP-0200695 (August 10, 1987)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 63152306 A	June 24, 1988		006	

APPLICATION-DATA:

PUB-NO	APPL-DATE	APPL-NO	DESCRIPTOR
JP63152306A	August 10, 1987	1987JP-0200695	

INT-CL (IPC): A01N 43/70; A01N 47/30; A01N 57/20; A01N 63/02

ABSTRACTED-PUB-NO: JP63152306A
BASIC-ABSTRACT:

A herbicide contains other herbicide of fosmidomycin or its salt, and ametryn or its salt or diuron. An amt. of the effective component is 5-1000 g/10 ares, pref. 100-500 g/10 ares. The mixing ratio of the fosmidomycin or its salt and the ametryn or its salt or diuron is 10:1-10:10, pref. 6:1-1:6.

The herbicide only may be used or the herbicide may be mixed with a disinfectant, nematicide, insecticide, plant growth regulator, fertiliser, or other herbicide.

The fosidomycin salt comprises pref. organic or inorganic base salt such as Na salt, K salt, or Ca salt. The ametryn salt comprises pref. organic or inorganic acid additive salt such as hydrochloride, sulphate, or phosphate. The ametryn and diuron comprises pref. 2-methyl mercapto-4-ethyl amino-6-isopropyl amino-s-triazine and 3-(3,4-dichlorophe nyl)-1,1-dimethyl urea.

USE/ADVANTAGE - The herbicide has good synergistic weeding action as compared with the herbicide having only one of the components. The herbicide is effective against e.g. broadleaved weeds, and Graminede weeds. The herbicide is used as dusting powder, granule, water-dispersible powder, liq. or emulsion by mixing the herbicide with various solid or liq. carriers such as e.g. kaolinite, and bentonite.

ABSTRACTED-PUB-NO: JP63152306A
EQUIVALENT-ABSTRACTS:

CHOSEN-DRAWING: Dwg.0/0

DERWENT-CLASS: C03
CPI-CODES: C05-B01P; C07-D13; C10-A13D; C12-C09; C12-P05;

WEST

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L1: Entry 1 of 1 File: JPAB May 24, 1986

PUB-NO: JP361106504A
DOCUMENT-IDENTIFIER: JP 61106504 A
TITLE: HERBICIDE

PUBN-DATE: May 24, 1986

INVENTOR-INFORMATION:
NAME COUNTRY
YAMAJI, TEIZO
AZUMA, SHIZUO
HIRAMATSU, TOSHIYUKI
ICHIKAWA, YATARO

ASSIGNEE-INFORMATION:
NAME COUNTRY
TEIJIN LTD

APPL-NO: JP59226873
APPL-DATE: October 30, 1984

INT-CL (IPC): A01N 57/02

ABSTRACT:

PURPOSE: To provide a herbicide containing a specific compound as a main active component, capable of killing a wide variety of weeds such as annual weeds and perennial weeds or suppressing the growth of the weeds by chlorosis, exhibiting excellent herbicidal activity and useful for the control of weeds in uncultivated land.

CONSTITUTION: The objective herbicide contains the N-substituted alkylamine phosphoric acid derivative of formula I (R1 is H, OH, lower alkyl or phenyl; R2 and R4 are H or lower alkyl; R3 is H or formyl; n is 1∼5), e.g. 3-(N-formyl-N-hydroxyamino)propylphosphonic acid isopropylamine salt, etc. as an active component. The compound of formula I wherein R1 is OH, R2 and R4 are H, R3 is formyl and n is 3 can be prepared e.g. by reacting the compound of formula II with the compound of formula III by Michaelis-Aransor reaction, reacting the resultant compound of formula IV with the compound of formula V to obtain the compound of formula VI, decomposing the compound with an acid, and finally reacting with formic acid in acetic anhydride to effect the N- formylation.